



## Effect on Coronary Artery Anatomy of Radiofrequency Catheter Ablation of Atrial Insertion Sites of Accessory Pathways

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**Objectives.** The purpose of this study was to analyze the effects of radiofrequency catheter ablation of the atrial insertion site of accessory pathways on the angiographic appearance of coronary arteries.

**Background.** Radiofrequency catheter ablation of accessory pathways requires the application of energy to the endocardial surface of the atrioventricular groove adjacent to the major epicardial coronary arteries. A systematic analysis of the effect of radiofrequency ablation on coronary arteries has not previously been demonstrated.

**Methods.** Seventy consecutive patients with 76 accessory pathways (7 right free wall, 44 left free wall, 12 posteroseptal, 8 antero-septal and 5 midseptal) were studied. Quantitative coronary

angiography was performed before, immediately after and a mean of  $69 \pm 42$  days after radiofrequency catheter ablation.

**Results.** Coronary artery diameter adjacent to the ablating electrode was  $2.6 \pm 0.9$  mm before ablation,  $2.7 \pm 0.9$  mm immediately after ablation and  $2.7 \pm 1.0$  mm at the time of follow-up study. Angiographic findings were unchanged from baseline in 69 of 70 patients immediately after ablation and in all 70 patients at the time of follow-up study.

**Conclusions.** Radiofrequency catheter ablation of the atrial insertion site of accessory pathways does not result in short-term angiographic changes in coronary artery anatomy.

(*J Am Coll Cardiol* 1993;21:1440-4)

Radiofrequency catheter ablation of an accessory pathway has been shown to be effective in the treatment of patients with the Wolff-Parkinson-White syndrome and orthodromic atrioventricular (AV) reentrant tachycardia using a concealed accessory pathway (1-8). This technique requires the delivery of radiofrequency energy to the endocardial surface of the AV groove adjacent to the major epicardial coronary arteries. The left circumflex coronary artery begins at the bifurcation of the left main coronary artery and travels down the epicardial surface of the left AV groove, supplying obtuse marginal branches to the posterolateral surface of the left ventricle. The right coronary artery follows the epicardial surface of the right AV groove and supplies the right ventricle and diaphragmatic surface of the left ventricle.

Despite the close proximity of the endocardial ablating electrode and the major epicardial coronary arteries, no previous study has demonstrated the effect of radiofrequency catheter ablation on coronary artery anatomy. Thus, it was the purpose of this study to analyze the effect of

radiofrequency catheter ablation on the angiographic appearance of coronary arteries.

### Methods

**Study patients.** The study group consisted of 70 consecutive patients referred for catheter ablation of an accessory AV pathway. There were 49 men and 21 women with a mean age of  $30 \pm 12$  years (range 10 to 57). Spontaneous arrhythmias included orthodromic AV reentrant tachycardia in 68 patients and atrial fibrillation with a rapid ventricular response in 18. These 70 patients had 76 accessory pathways (44 left free wall, 7 right free wall, 12 posteroseptal, 8 antero-septal and 5 midseptal). Fifty-eight accessory pathways were manifest and 18 were concealed.

**Electrophysiologic study.** After giving written informed consent, patients were studied in the postabsorptive state under moderate sedation. Electrophysiologic testing was performed to localize the accessory pathway and to characterize its functional properties. Electrode catheters introduced through the femoral and subclavian veins were positioned in the high right atrium, His bundle position, right ventricular apex and coronary sinus. This study consisted of standard atrial and ventricular extrastimulus techniques, atrial and ventricular ramp pacing and induction of atrial fibrillation.

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Manuscript received July 13, 1992; revised manuscript received October 8, 1992; accepted October 28, 1992.

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**Accessory pathway localization.** Initial epicardial localization of accessory pathways was achieved by coronary sinus mapping for left-sided accessory pathways and right coronary artery mapping for selected right-sided accessory pathways. Septal pathways were localized with endocardial mapping only.

Endocardial localization and ablation of all left-sided accessory pathways was accomplished using a transseptal approach. A modified Bruckebrough method was used for transseptal catheterization in patients without a patent foramen ovale (1). Endocardial localization and ablation of right free wall and septal accessory pathways was accomplished using endocardial mapping of the right AV groove from the right femoral vein. A modified transseptal sheath was often used for catheter stabilization for right-sided pathways as well. Endocardial mapping was performed using a steerable 7F 3- or 4-mm tip electrode catheter with 2-mm interelectrode spacing (Mansfield-Webster, Boston Scientific). Bipolar (30- to 500-Hz bandpass filter) and distal electrode unipolar (0.01- to 500-Hz bandpass filter) electrograms were continuously recorded from the ablation catheter. The atrial insertion sites of accessory pathways were localized during sinus rhythm by identifying an AV ratio  $\geq 1$  on the bipolar electrogram and identifying PR segment deviation on the unipolar recording.

Accessory pathways were localized using both antegrade and retrograde evaluation. Initial localization was achieved by mapping of the shortest surface QRS complex to local atrial activation in orthodromic AV reentrant tachycardia or right ventricular pacing stimulus to local atrial activation during ventricular pacing. Retrograde localization was also achieved by following local ventriculoatrial conduction times. Antegrade localization involved the shortest AV conduction time or interval from the local ventricular electrogram to onset of the delta wave.

**Ablation procedure.** Radiofrequency energy was delivered as an unmodulated 500-kHz alternating current from a standard electrosurgical unit (System 5000, C.R. Bard). Energy was delivered in a monopolar mode between the ablating electrode and a large surface area skin electrode (R2 Cath-Pads, Darox).

**Coronary angiography.** Coronary angiography was performed in all patients in standard 30° right anterior oblique and left anterior oblique projections using small amounts of hand-injected contrast medium. Angiograms were obtained at the beginning of the study, after successful ablation and at the time of the follow-up study. Angiograms were analyzed by two independent observers who were not aware of the sequence of the studies.

**Quantitative angiography.** Quantitative angiography was performed on all coronary arteries adjacent to sites of radiofrequency energy delivery. The system used was the CardioTrace Cardiac Evaluation System (version 1.5, Cine Graphics), which has been previously validated against a reference system (9). This allows for measurement of the lumen diameter by a geometric mode, which employs an

edge detection algorithm, and a densitometric mode. Absolute vessel diameter was then calculated by comparison with a reference angiographic catheter.

**Associated tests.** All patients also underwent two-dimensional echocardiography before and on the day after radiofrequency catheter ablation. These were analyzed by an independent observer, assessing for wall motion abnormalities.

Subjects also had serial measurements of creatine kinase with MB isoenzymes to evaluate for enzymatic evidence of myocardial necrosis. These data were obtained every 6 h for a total of 24 h.

**Statistical analysis.** All results are reported as the mean value  $\pm 1$  SD. The data were analyzed using repeated measures analysis of variance.

## Results

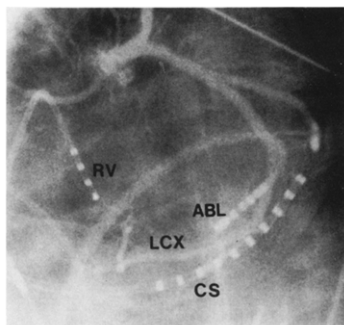
**Ablation variables.** The delivery of radiofrequency energy resulted in the elimination of accessory pathway conduction in 71 (93%) of 76 accessory pathways. Patients required  $6.3 \pm 5.1$  (range 1 to 23) applications of radiofrequency energy. The mean cumulative energy level was  $2,341 \pm 2,233$  J/patient. At the site of successful ablation, a mean radiofrequency power of  $25.7 \pm 2.6$  W was delivered for mean of  $21.0 \pm 8.9$  s. The mean total radiofrequency energy delivered was  $546 \pm 252$  J at the site of successful ablation. The overall time of the ablation procedure, including diagnostic electrophysiologic study, ablation and coronary angiography, was  $4.9 \pm 2.3$  h. Fluoroscopy time was  $62.6 \pm 47$  min.

The five unsuccessful ablation attempts were performed on two left free wall, one septal and two right free wall accessory pathways. Three of these patients later underwent surgical ablation, and two are being treated medically. Two of the patients with unsuccessful ablation attempts had two accessory pathways at baseline.

**Catheter positioning.** The ablating electrode was positioned 2 to 10 mm from the coronary artery. This distance was measured using the 7F (2.3 mm) ablation catheter as a reference catheter. The maximal distance of the ablating electrode to the closest segment of coronary artery, in orthogonal views, was taken as the actual distance.

Figure 1 demonstrates the typical angiographic appearance of catheters approaching a left free wall accessory pathway, as seen in a left anterior oblique projection with caudal angulation. The ablation catheter crosses the interatrial septum and parallels the course of the coronary sinus catheter. In this view, the left circumflex coronary artery is located in close proximity to the ablating electrode and the coronary sinus catheter. Accessory pathways occur at any point along the AV groove and thus are adjacent to the left circumflex coronary artery along any portion of its course.

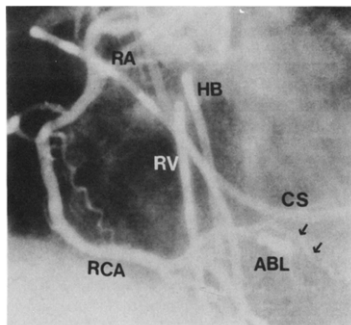
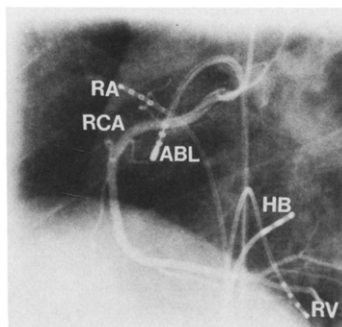
Figure 2 represents the angiographic appearance of catheter positioning for a right free wall accessory pathway in a left anterior oblique projection. The ablating electrode is



**Figure 1.** Postablation angiogram in the left anterior oblique projection demonstrating catheter positions to approach a left free wall accessory pathway. The distal electrode of the ablation catheter (ABL) crosses the interatrial septum and parallels the course of the coronary sinus catheter (CS). The left circumflex coronary artery (LCX) is located near the ablating electrode. RV = right ventricular catheter.

located on the endocardial surface of the right AV groove in close proximity to the right coronary artery. The ablating electrode can be positioned anywhere along the course of the right coronary artery depending on the location of the accessory pathway.

**Figure 2.** Postablation angiogram in the left anterior oblique projection demonstrating catheter positions to approach a right free wall accessory pathway. The ablating electrode (ABL) is localized on the endocardial surface of the right AV groove in close proximity to the right coronary artery (RCA). HB = His bundle catheter; RA = right atrial catheter; RV = right ventricular catheter.



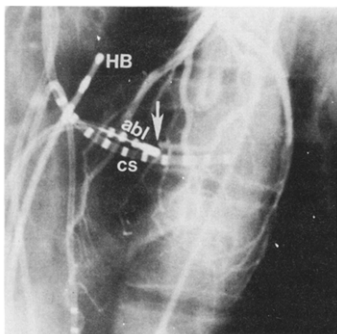
**Figure 3.** Postablation angiogram in the left anterior oblique projection demonstrating catheter positions to approach a posteroseptal accessory pathway. The ablating electrode (ABL) is located near a posterolateral branch (black arrows) of the right coronary artery (RCA). Abbreviations as in Figures 1 and 2.

**Figure 3** is a left anterior oblique projection demonstrating ablation of a posteroseptal accessory pathway. The ablating electrode is positioned within the coronary sinus os. In this view, a posterolateral branch of the right coronary artery passes between the coronary sinus catheter and the ablating electrode. With a right dominant coronary circulation, the ablating electrode lies adjacent to a posterolateral branch of the right coronary artery, as in **Figure 3**. However, in a left dominant circulation the ablating electrode is in close proximity to the distal left circumflex coronary artery. The approach to posteroseptal accessory pathways often requires entering the coronary sinus os. Efforts are made to stay within the os only and to avoid entering the true coronary sinus, which is an epicardial structure.

**Quantitative angiography.** Quantitative angiography was performed on all 76 coronary artery segments, which were adjacent to the ablating electrode, at the site of successful radiofrequency energy delivery. No significant changes in the mean lumen diameter occurred at any point in the study period. The mean segmental coronary artery diameter was  $2.6 \pm 0.9$  mm before ablation,  $2.7 \pm 0.9$  mm immediately after ablation and  $2.7 \pm 1.0$  mm at  $69 \pm 42$  days of follow-up ( $p = \text{NS}$ ).

There were no coronary artery stenoses  $>50\%$  of lumen diameter in any baseline coronary angiogram. Immediately after ablation, there was no significant change in coronary artery diameter in 69 of 70 patients.

One patient demonstrated a change in coronary anatomy immediately after ablation of a left posteroseptal accessory pathway. In contrast to the procedure in our other patients, in this patient the ablating electrode was positioned within



**Figure 4.** Postablation angiogram in the left anterior oblique projection demonstrating an occlusion (white arrow) of a distal branch of the left circumflex coronary artery at the site of the ablating electrode (abl). The ablating electrode was positioned within the coronary sinus (cs). HB = His bundle catheter.

the coronary sinus and in close proximity to a small distal branch of a left dominant left circumflex coronary artery. Figure 4 shows an angiographic frame in the left anterior oblique view that was obtained from this patient immediately after a successful ablation procedure using 25 W of radiofrequency energy for 20 s. The study reveals occlusion of a distal branch of the left circumflex coronary artery at the site of the ablating electrode. As a result, however, this patient developed no chest pain, elevation of serum creatine kinase levels or echocardiographic wall motion abnormalities. She was treated empirically with nifedipine and had normal findings on a 6-week follow-up coronary angiogram that demonstrated no significant change from the baseline study before ablation. Thus, coronary artery spasm was believed to be the origin of the acute vessel closure in this patient.

**Associated tests.** Analysis of the echocardiograms revealed no differences after radiofrequency catheter ablation. Specifically, there were no new wall motion abnormalities or changes in tricuspid or mitral valve function. Serial measurements of creatine kinase with MB isoenzymes revealed no evidence of myocardial necrosis.

## Discussion

Although radiofrequency catheter ablation of the atrial insertion site of accessory pathways involves the application of energy to the endocardial surface of the AV groove, the effect on the major epicardial coronary arteries has not been previously demonstrated. In the present study, quantitative coronary angiography documented that radiofrequency endocardial ablation did not result in angiographic changes in

coronary artery anatomy either immediately or on follow-up examination.

**Ablation within the coronary sinus.** In the one patient whose coronary artery anatomy did change, energy was delivered within a posterior descending coronary vein of the coronary sinus and to the epicardial surface of the heart rather than to the endocardial location used in other patients. The lesion was presumed to be a result of coronary artery spasm because subsequent angiographic findings were normal, and the patient developed no evidence of myocardial necrosis.

Delivery of radiofrequency energy within the coronary sinus has frequently been reported to result in complications. Direct current ablation has been reported to lead to barotraumatic coronary sinus rupture, often requiring surgical intervention and to myocardial infarction caused by spasm of a branch of the right coronary artery (10-12). Radiofrequency ablation has also resulted in hemopericardium and cardiac tamponade when radiofrequency current was delivered in a small venous branch of the coronary sinus (2).

**Ablation from the endocardial surface.** Catheter ablation outside of the coronary sinus has also been reported to cause coronary artery injury. A subtotal occlusion of the right posterior descending coronary artery and a high grade narrowing of a posterolateral branch of the right coronary artery were observed after endocardial direct current ablation of a right-sided accessory pathway (13). There has been at least one report of endocardial radiofrequency catheter ablation resulting in spasm of the left circumflex coronary artery (14). Inadvertent positioning of the ablating electrode within the coronary artery itself has also been reported to result in coronary obstruction (3,15).

Although injuries to the coronary tree and coronary sinus have been reported, no previous study has provided a systematic analysis of a large consecutive series of accessory pathway ablations in multiple locations. Previously, small series have failed to demonstrate angiographic abnormalities after catheter ablation. Coronary angiography was performed 4 to 6 months after direct current ablation of posteroseptal accessory pathways in 10 patients and revealed no coronary artery abnormalities (11). Also, direct current ablation of the atrial insertion site of three posteroseptal, one left free wall and two right free wall accessory pathways did not result in abnormalities on coronary angiography (16). Van Hare et al. (5) demonstrated integrity of the right coronary artery in three pediatric patients after radiofrequency ablation.

In our series, radiofrequency catheter ablation of accessory pathways did not result in angiographic coronary artery abnormalities despite the delivery of current from the ablating electrode to the endocardial surface of the AV groove. The effects of radiofrequency current are due to an increase in temperature and tissue desiccation (17). This results in a well demarcated area of coagulation necrosis within the myocardium. In many instances energy was delivered in

close proximity to a coronary artery, and there was concern that the temperature could result in coronary artery injury. However, the continuous flow of blood through the coronary arteries may result in convective heat loss and thus protect the coronary arteries from thermal damage (18).

**Limitations of the study.** Our study has two important limitations. 1) The follow-up period (mean  $69 \pm 42$  days). Although no significant changes in coronary artery anatomy were seen during this period, it is possible that angiographic changes would not be observed this early. Thus, repeat angiography after several years would be helpful. 2) We used coronary angiography to study coronary artery anatomy. Although coronary angiography is the standard for such study, it may miss some histologic or functional coronary lesions. Direct current shocks within the coronary sinus of dogs have resulted in mild to marked intimal hyperplasia despite normal coronary angiographic findings (12). Radiofrequency catheter ablation within the coronary sinus has also been shown to result in necrotizing arteritis and arterial sclerosis (19). However, histologic evidence of coronary artery lesions has not been observed with endocardial radiofrequency catheter ablation.

**Conclusions.** Our results demonstrate that radiofrequency catheter ablation of the atrial insertion site of accessory pathways does not result in angiographic coronary artery abnormalities. Routine coronary angiography is not necessary after this procedure.

We thank Nona Shepard for skillful preparation of this manuscript.

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